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Derwent World Patents Index
IBM Technical Disclosure Bulletins

Term: L1 and ischemia

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DATE: Wednesday, June 28, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB; PLUR=YES; OP=ADJ			
L2	L1 and ischemia	18	L2
L1	Mk2 and antisense	114	L1

END OF SEARCH HISTORY

MAPK
MAPK-2
MK-2
MAP KINASE ACTIVATED PROTEIN KINASE

} & ANTISENSE

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NEWS 6	MAY 11	KOREAPAT updates resume
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NEWS 8	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2
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NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006. V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT http://download.cas.org/express/v8.0-Discover/

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FILE 'HOME' ENTERED AT 15:59:36 ON 28 JUN 2006

=> file medline embase biosis scisearch caplus biotechno

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 16:00:13 ON 28 JUN 2006

FILE 'EMBASE' ENTERED AT 16:00:13 ON 28 JUN 2006

FILE 'BIOSIS' ENTERED AT 16:00:13 ON 28 JUN 2006

FILE 'SCISEARCH' ENTERED AT 16:00:13 ON 28 JUN 2006

FILE 'CAPLUS' ENTERED AT 16:00:13 ON 28 JUN 2006
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FILE 'BIOTECHNO' ENTERED AT 16:00:13 ON 28 JUN 2006


```
=> s map kinase-activated protein kinase
L1         371 MAP KINASE-ACTIVATED PROTEIN KINASE

=> s mk2
L2        2187 MK2

=> s mk-2
L3         892 MK-2

=> s antisense
L4      157768 ANTISENSE

=> s l1 and l2
L5          48 L1 AND L2

=> s l1 and l4
L6           1 L1 AND L4

=> s l2 and l4
L7           1 L2 AND L4

=> s l3 and l4
L8           6 L3 AND L4

=> d l6

L6   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   2004:182659  CAPLUS
DN   140:229441
TI   Methods of reducing ischemic injury with compounds reducing activity of
     MAP kinase-activated protein
     kinase 2
IN   Wang, Xinkang; Schieven, Gary; Feuerstein, Giora Z.
PA   Bristol-Myers Squibb Company, USA
SO   PCT Int. Appl., 39 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
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PI   WO 2004017909        A2    20040304    WO 2003-US26337         20030821
     WO 2004017909        A3    20040819
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             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY; TJ, TM, TN,
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            FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
            BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2003259991        A1    20040311    AU 2003-259991          20030821
     US 2004110710        A1    20040610    US 2003-645190          20030821
     EP 1546181           A2    20050629    EP 2003-793290          20030821
        R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     JP 2006502142        T2    20060119    JP 2004-529851          20030821
PRAI US 2002-405586P      P     20020823
     WO 2003-US26337      W     20030821


=> d l7

L7   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2006 ACS on STN
AN   2004:182659  CAPLUS
DN   140:229441
TI   Methods of reducing ischemic injury with compounds reducing activity of
     MAP kinase-activated protein kinase 2
IN   Wang, Xinkang; Schieven, Gary; Feuerstein, Giora Z.
PA   Bristol-Myers Squibb Company, USA
SO   PCT Int. Appl., 39 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
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PI   WO 2004017909        A2     20040304    WO 2003-US26337         20030821
     WO 2004017909        A3     20040819
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            GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
            LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
            PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
            TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
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            FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
            BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2003259991        A1    20040311    AU 2003-259991          20030821
     US 2004110710        A1    20040610    US 2003-645190          20030821
     EP 1546181           A2    20050629    EP 2003-793290          20030821
        R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     JP 2006502142        T2    20060119    JP 2004-529851          20030821
PRAI US 2002-405586P      P     20020823
     WO 2003-US26337      W     20030821


=> d ti 1-6 l8

L8   ANSWER 1 OF 6     MEDLINE on STN
TI   Phorbol ester-induced G1 arrest in BALB/MK-2 mouse
     keratinocytes is mediated by delta and eta isoforms of protein kinase C.

L8   ANSWER 2 OF 6  EMBASE  COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN
TI   Phorbol ester-induced G1 arrest in BALB/MK-2 mouse
     keratinocytes is mediated by δ and η isoforms of protein kinase
     C.
```

L8 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Phorbol ester-induced G1 arrest in BALB/**MK-2** mouse keratinocytes is mediated by delta and eta isoforms of protein kinase C.

L8 ANSWER 4 OF 6 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Phorbol ester-induced G1 arrest in BALB/**MK-2** mouse keratinocytes is mediated by delta and eta isoforms of protein kinase C

L8 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Phorbol ester-induced G1 arrest in BALB/**MK-2** mouse keratinocytes is mediated by δ and η isoforms of protein kinase C

L8 ANSWER 6 OF 6 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
TI Phorbol ester-induced G1 arrest in BALB/**MK-2** mouse keratinocytes is mediated by δ and η isoforms of protein kinase C

=> s l1 and ischemia
L9 13 L1 AND ISCHEMIA

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 8 DUP REM L9 (5 DUPLICATES REMOVED)

=> d ti 1-8

L10 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI DEF domain-containing members of the MAP kinase pathway and their use in screening for drug inhibitors

L10 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Role of F-actin organization in p38 MAP kinase-mediated apoptosis and necrosis in neonatal rat cardiomyocytes subjected to simulated **ischemia** and reoxygenation

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Methods of reducing ischemic injury with compounds reducing activity of **MAP kinase-activated protein kinase** 2

L10 ANSWER 4 OF 8 MEDLINE on STN DUPLICATE 1
TI Mitogen-activated protein kinase-activated protein (MAPKAP) kinase 2 deficiency protects brain from ischemic injury in mice.

L10 ANSWER 5 OF 8 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Stimulation of multiple MAPK pathways by mechanical overload in the perfused amphibian heart

L10 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Ischemic preconditioning triggers tyrosine kinase signaling: a potential role for MAPKAP kinase 2

L10 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Ischemic preconditioning: role of multiple kinases in signal amplification and modulation

L10 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Involvement of a tyrosine kinase-dependent signal transduction process involving p-38 MAP kinases and MAPKAP kinase 2 in ischemic

preconditioning.

=> d ab 1 3 4 6 8 l10

L10 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
AB Mitogen-activated protein (MAP) kinases (e.g., ERK1/2) phosphorylate a variety of target proteins including, for example, several immediate-early gene products (e.g., Fos, Myc, and Jun family proteins). Certain phosphorylation reactions require binding of the MAP kinase to the DEF domain of the target protein. Inhibitors that block this interaction may be useful therapeutics for human disease, including as antineoplastic agents. This invention provides several advantages over known therapies that directly target the MAP kinase signaling cascade. Typically, most compds. that inhibit the MAP kinase pathway are non-specific and inhibit more than one enzyme, and the targeted inhibited kinases are not available to perform normal physiol. functions necessary for cell survival, whereas therapeutic methods of the present invention inhibit the activation of particular target proteins and leave the MAP kinases enzymically active and available to phosphorylate other non-DEF domain-containing proteins. Thus, DEF domains are identified in a large number of proteins, and the principles of the invention are exemplified using the immediate-early gene, c-Fos. Screening assays useful for identifying compds. that inhibit the MAP kinase-DEF domain interaction are also disclosed.

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
AB The present invention includes methods of reducing the activity, such as enzymic activity and expression, of mitogen-activated protein (**MAP**) **kinase-activated protein kinase** 2 (MK2). The present invention further includes methods for identifying compds. useful for reducing such activity, and methods for reducing ischemic injury by the administration of such compds. Ischemic brain injury was significantly reduced in MK2 deficient mice compared to theat of wild type mice following either transient or permanent occlusion of the middle cerebral artery.

L10 ANSWER 4 OF 8 MEDLINE on STN DUPLICATE 1
AB Mitogen-activated protein (**MAP**) **kinase-activated protein kinase** 2 (MK2) is one of several kinases directly regulated by p38 MAP kinase. A role of p38 MAP kinase in ischemic brain injury has been previously suggested by pharmacological means. In the present study, we provide evidence for a role of MK2 in cerebral ischemic injury using MK2-deficient (MK2(-/-)) mice. MK2(-/-) mice subjected to focal **ischemia** markedly reduced infarct size by 64 and 76% after transient and permanent **ischemia**, respectively, compared with wild-type mice. Furthermore, MK2(-/-) mice had significant reduction in neurological deficits. Real-time PCR analysis identified a significantly lower expression in interleukin-1beta mRNA (53% reduction) but not in tumor necrosis factor-alpha mRNA in MK2(-/-) mice over wild-type animals after ischemic injury. The significant reduction in interleukin-1beta was also confirmed in MK2(-/-) mice by enzyme-linked immunosorbent assay. The marked neuroprotection from ischemic brain injury in MK2(-/-) mice was not associated with the alteration of hemodynamic or systemic variables, activation of caspase-3, or apoptosis. Our data provide new evidence for the involvement of MAP kinase pathway in focal ischemic brain injury and suggest that this effect might be associated with the expression of interleukin-1beta in the ischemic brain tissue.

L10 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
AB Myocardial adaptation to **ischemia** has been shown to activate protein tyrosine kinase, potentiating activation of phospholipase D, which leads to the stimulation of mitogen-activated protein (MAP) kinases and MAP kinase-activated protein (MAPKAP) kinase 2. The present study sought

to further examine the signal transduction pathway for the MAPKAP kinase 2 activation during ischemic adaptation. Isolated perfused rat hearts were adapted to ischemic stress by repeated **ischemia** and reperfusion. Hearts were pretreated with genistein to block tyrosine kinase, whereas SB-203580 was used to inhibit p38 MAP kinases. Western blot anal. demonstrated that p38 MAP kinase is phosphorylated during ischemic stress adaptation. Phosphorylation of p38 MAP kinase was blocked by genistein, suggesting that activation of p38 MAP kinase during ischemic adaptation is mediated by a tyrosine kinase signaling pathway. MAPKAP kinase 2 was estimated by following in vitro phosphorylation with recombinant human heat shock protein 27 as specific substrate for MAPKAP kinase 2. Again, both genistein and SB-203580 blocked the activation of MAPKAP kinase 2 during myocardial adaptation to **ischemia**. Immunofluorescence microscopy with anti-p38-antibody revealed that p38 MAP kinase is primarily localized in perinuclear regions. P38 MAP kinase moves to the nucleus after ischemic stress adaptation. After **ischemia** and reperfusion, cytoplasmic striations in the myocytes become obvious, indicating translocation of p38 MAP kinase from nucleus to cytoplasm. Corroborating these results, myocardial adaptation to **ischemia** improved the left ventricular functions and reduced myocardial infarction that were reversed by blocking either tyrosine kinase or p38 MAP kinase. These results demonstrate that myocardial adaptation to **ischemia** triggers a tyrosine kinase-regulated signaling pathway, leading to the translocation and activation of p38 MAP kinase and implicating a role for MAPKAP kinase 2.

L10 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

=> d 1 3 4 6 8 l10

L10 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:71066 CAPLUS
DN 142:170050
TI DEF domain-containing members of the MAP kinase pathway and their use in screening for drug inhibitors
IN Blenis, John; Murphy, Leon O.
PA President and Fellows of Harvard College, USA
SO PCT Int. Appl., 104 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005007090	A2	20050127	WO 2004-US21514	20040702
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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PRAI	US 2003-484761P	P	20030703		

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:182659 CAPLUS
DN 140:229441
TI Methods of reducing ischemic injury with compounds reducing activity of **MAP kinase-activated protein**

kinase 2
IN Wang, Xinkang; Schieven, Gary; Feuerstein, Giora Z.
PA Bristol-Myers Squibb Company, USA
SO PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004017909	A2	20040304	WO 2003-US26337	20030821
	WO 2004017909	A3	20040819		
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	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003259991	A1	20040311	AU 2003-259991	20030821
	US 2004110710	A1	20040610	US 2003-645190	20030821
	EP 1546181	A2	20050629	EP 2003-793290	20030821
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	JP 2006502142	T2	20060119	JP 2004-529851	20030821
PRAI	US 2002-405586P	P	20020823		
	WO 2003-US26337	W	20030821		

L10 ANSWER 4 OF 8 MEDLINE on STN DUPLICATE 1
AN 2002666141 MEDLINE
DN PubMed ID: 12215446
TI Mitogen-activated protein kinase-activated protein (MAPKAP) kinase 2 deficiency protects brain from ischemic injury in mice.
AU Wang Xinkang; Xu Lin; Wang Hugh; Young Peter R; Gaestel Matthias; Feuerstein Giora Z
CS Department of Cardiovascular Sciences, Bristol-Myers Squibb Company, Wilmington, Delaware 19880-0400, USA.. xinkang.wang@bms.com
SO The Journal of biological chemistry, (2002 Nov 15) Vol. 277, No. 46, pp. 43968-72. Electronic Publication: 2002-09-04.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200301
ED Entered STN: 13 Nov 2002
Last Updated on STN: 3 Jan 2003
Entered Medline: 2 Jan 2003

L10 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
AN 1998:759299 CAPLUS
DN 130:123195
TI Ischemic preconditioning triggers tyrosine kinase signaling: a potential role for MAPKAP kinase 2
AU Maulik, Nilanjana; Yoshida, Tetsuya; Zu, You-Li; Sato, Motoaki; Banerjee, Anirban; Das, Dipak K.
CS Departments of Surgery and Physiology, University of Connecticut School of Medicine, Farmington, CT, 06030-1110, USA
SO American Journal of Physiology (1998), 275(5, Pt. 2), H1857-H1864
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society

DT Journal
LA English
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1997:424888 BIOSIS
DN PREV199799724091
TI Involvement of a tyrosine kinase-dependent signal transduction process involving p-38 MAP kinases and MAPKAP kinase 2 in ischemic preconditioning.
AU Das, Dipak K.
CS Univ. Connecticut Sch. Med., Farmington, CT, USA
SO Journal of Molecular and Cellular Cardiology, (1997) Vol. 29, No. 7, pp. A272.
Meeting Info.: XIX Annual Meeting of the International Society for Heart Research (American Section) Cardiovascular Injury, Repair and Adaptation. Vancouver, British Columbia, Canada. July 23-27, 1997.
CODEN: JMCDAY. ISSN: 0022-2828.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 8 Oct 1997
Last Updated on STN: 21 Nov 1997

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	65.55	65.76

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.25	-2.25

FILE 'STNGUIDE' ENTERED AT 16:17:06 ON 28 JUN 2006

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jun 23, 2006 (20060623/UP).